Changes in serum lipid concentrations during first 24 hours after myocardial infarction

The serum total cholesterol concentration falls after myocardial infarction, reaching its lowest value between the sixth and ninth days. Two reports suggested, however, that total cholesterol concentrations measured in the first 24-48 hours after myocardial infarction are probably close to pre-infarction values. Both studies assumed that the concentrations measured months before or after myocardial infarction accurately reflect the concentrations at infarction, but this may not be valid, particularly with the increasing use of dietary intervention in patients with symptoms of heart disease.

Although total cholesterol concentration cannot be measured in the hours before myocardial infarction, documentation of any changes in the early hours after infarction is likely to enable a more accurate estimate of pre-infarction concentrations to be made. We present data on changes in serum lipid concentrations during the first 24 hours after myocardial infarction in patients admitted to hospital within four hours after the onset of chest pain.

Patients, methods, and results

Non-fasting total serum cholesterol and high density lipoprotein cholesterol concentrations were measured immediately after admission to hospital and at eight, 16, 24, and 48 hours after admission in 25 patients with myocardial infarction admitted to Auckland coronary care units during 1985 within four hours after the onset of chest pain. The patients comprised 22 men and three women aged 35-69 (mean age 57.8 years). The mean time of the first sample was 2 hours 58 minutes after the onset of chest pain (range 1 hour 35 minutes to 3 hours 55 minutes).

Lipid estimations were done in a laboratory standardised according to the cholesterol standardisation programme at the Centers for Disease Control, Atlanta, Georgia. The between batch coefficient of variation for total cholesterol was 0-4% at 6.35 mmol/l, for high density lipoprotein cholesterol it was approximately 6% at 1.00 mmol/l. Paired t tests were used to assess the significance of differences between lipid concentrations. The level of significance was set at 5% (two sided). The study was designed to detect a 10% change in the concentrations of total cholesterol and high density lipoprotein cholesterol from 6.2 and 1.0 mmol/l, respectively, with a type II error of 0-05.

Eight hours after admission the total cholesterol concentration had fallen by 1-8% from the value on admission; at 16 hours it had fallen by 2-0%, at 24 hours by 6-6%, and at 48 hours by 15-8% (figure). There were no significant differences between the concentrations on admission and those at eight and 16 hours. At 24 hours the difference was significant (p=0.004) but remained small (4 mmol/l). A simple linear regression model was fitted to the first four data points for each patient with time 0 corresponding to the onset of chest pain. A test for the homogeneity of the slopes between patients showed no significant differences, and the estimated common slope of the fitted model was 0.0166 mmol/l/h.

Eight hours after admission there was a significant increase (p=0.025) in the concentration of high density lipoprotein cholesterol of 6-9% (figure). The subsequent measurements at 16, 24, and 48 hours showed almost no change from this value.

**Results of study**

<table>
<thead>
<tr>
<th>Glaucomatous discs (n=16)</th>
<th>Consultants (n=20)</th>
<th>Junior doctors (n=16)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Correct (ie, specificity)</td>
<td>7 (59)</td>
<td>8 (53)</td>
</tr>
<tr>
<td>Incorrect (false positive)</td>
<td>2 (15)</td>
<td>1 (9)</td>
</tr>
<tr>
<td>Scores on individual discs (no of discs)</td>
<td>6 (6) (41)</td>
<td>6 (6) (38)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Non-glaucomatous discs (n=27)</th>
<th>Consultants (n=20)</th>
<th>Junior doctors (n=16)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Correct (ie, specificity)</td>
<td>20 (77)</td>
<td>18 (69)</td>
</tr>
<tr>
<td>Incorrect (false positive)</td>
<td>2 (9)</td>
<td>3 (13)</td>
</tr>
<tr>
<td>Scores on individual discs (no of discs)</td>
<td>3 (8) (14)</td>
<td>5 (0) (18)</td>
</tr>
</tbody>
</table>

We are grateful to Dr David Appleton for his help with statistical analysis.


Department of Ophthalmology, Newcastle General Hospital, Newcastle upon Tyne NE4 6BE
CHRISTOPHER M WOOD, FRCS, senior registrar
ROBIN C BOSANQUET, FRCS, consultant
Correspondence to: Mr Wood.

Trends in concentrations of total cholesterol and high density lipoprotein cholesterol after myocardial infarction. Values are means (SD).

Comment

Our study confirms previous reports that the serum total cholesterol concentration in the first 24 hours after infarction is likely to reflect preinfarction concentrations. The relative change in the high density lipoprotein cholesterol concentration after myocardial infarction was greater than that of the total cholesterol concentration during this period, but this is probably of little clinical importance. As most patients with myocardial infarction are admitted within 24 hours after the onset of chest pain, and many patients are likely to miss screening scheduled for after their
Severe rectal bleeding due to Salmonella paratyphi B

Lifethreatening rectal haemorrhage is rare. The commonest causes are diverticular disease, colonic angiodysplasia, Meckel’s diverticulum, peptic ulceration in the stomach or duodenum, and trauma. We present a case of massive colonic and rectal bleeding due to Salmonella paratyphi B that required emergency colectomy.

Case report
An 18 year old girl presented with a two hour history of bleeding from the rectum. This followed a week illness similar to flu, four days of constipation, and then diarrhoea. There was no relevant medical or drug history. On admission she was shocked and febrile (39-6°C) with pulse 120 beats/min and blood pressure 100/60 mm Hg. There were no abnormal signs, but rectal examination showed fresh clots of blood.

Despite active resuscitation her condition remained critical. Her haemoglobin concentration was 40 g/l, but all other tests including coagulation studies yielded normal results. Findings on gastroscopy were normal. Superior and inferior mesenteric arteriograms showed no evidence of bleeding. Colonoscopy was unsuccessful because of the profuse haemorrhage. A scan using red cells tagged with technetium-99m showed pooling of blood only in the colon, and at laparotomy only the left colon was affected. Colotomy showed bleeding from innumerable shallow ulcers. Extended left hemicolectomy and end colostomy were performed. Continued bleeding from the ulcerated mucosa in the rectal stump required an underrunning suture and packing of the rectum. She received a total of 26 litres of blood and plasma.

Culture of a stool specimen showed Salmonella paratyphi B (phage type Dunde). There were innumerable superficial ulcers 1-12 mm in diameter in the colon, their severity increasing distally (figure). Histologically the ulcers were characterised by a paucity of neutrophils in the granulation tissue and exudate. The colonic lymphoid aggregates contained many plump histiocytes, some containing debris (“typhoid cells”); similar cells were present in the dilated sinuses of the draining lymph nodes. No vasculitis was seen.

Postoperatively no further complications occurred. The rectal pack was removed after 72 hours and chloramphenicol 50 mg/kg given for 10 days. The colostomy was closed uneventfully two months later.

Comment
Paratyphoid B is the commonest enteric fever in western Europe and is more commonly acquired by ingestion of infected food than contaminated water. Postoperatively this patient said that she had eaten a hamburger in a seaside resort two weeks before her admission; this incubation time is consistent with paratyphoid fever.

The methods available for identifying sites of bleeding in the gastrointestinal tract have limitations. Angiography failed to identify the nature of the colonic and rectal haemorrhage in this case, which highlights its inadequacy when there are multiple bleeding sites that individually bleed less than 3 ml per minute, but together produce extensive blood loss. The usefulness of scanning with red cells labelled with technetium in detecting the site of bleeding can be limited by blood background activity as there must be a sufficient difference between the levels of circulating and extravasated tracer for the bleeding to be localised reliably. It is important to exclude a cause in the upper gastrointestinal tract by endoscopy. Preoperative colonoscopy in these cases, however, is difficult because of the large amounts of clotted blood in unprepared bowel. In retrospect perioperative colonoscopy after colonic lavage might have provided valuable information in this case and is a useful investigation in massive colonic haemorrhage.

Ulceration of the gastrointestinal tract in paratyphoid fever is usually limited to the terminal ileum, sometimes leading to the passage of traces of blood through the rectum and even severe bleeding. Intestinal haemorrhage is a rare complication, occurring in 1% of cases. We can find no reports of a case similar to ours.

We thank Mr Max Rendall for permission to report this case.


Department of Surgery, Guy's Hospital, London SE1 9RT
J A SPENCE, BSc, MB, house surgeon
R MOGERE, FRCS, surgical registrar
T J PALMER, MRCP, MRCPATH, senior registrar in clinical microscopy
P H ROWE, MA, FRCS, senior surgical registrar

Correspondence to: Mr Rowe.